Editorials

Physicians as 'Double Agents'

IN A RECENT COLUMN in *American Medical News,* Harry Schwartz pointed out that physicians were being forced more and more to become what amounts to "double agents" in patient care, with all that this implies. A double agent in the world of international intrigue agrees to work on behalf of two masters who have conflicting goals and purposes. Often, neither is aware of the agent's responsibility to the other. In addition, it is sometimes necessary for double agents to act on behalf of only themselves, for their own well-being or even survival. The role of a double agent has to be an uncomfortable one, especially for physicians. By definition a double agent can hardly be loyal to both masters and perhaps to neither of them, and, in any event, loyalty to oneself may sometimes become paramount.

Many physicians—and there are more each day—are finding themselves in this uncomfortable role. On the one hand, patients and the public expect them to work fully on behalf of those patients who seek their help, while at the same time they are being pressured more and more to serve as agents of government and others who are trying to reduce the amount and cost of services that are purchased by third parties for patients. This unwanted dual role is already causing serious discomfort for many physicians, who, caught in what seems like an ever-tightening vise, must also try to protect their own personal moral integrity and sometimes their own economic survival. If nothing is done, all this can only get worse as the demand for services inevitably increases in the face of an obviously growing need to curtail costs.

There are those who say that the supply of resources would be adequate to meet the demand for services if ineffective and unnecessary services were eliminated, and they call upon the medical profession to help bring this about. At best, however, and even if fully accomplished, this would do only for a while because the disparity between a virtually unlimited demand for services and clearly limited resources can only increase. In the meantime, many who are powerfully situated believe that the cost control measures that are being imposed, including putting physicians in the position of "gatekeeper," with responsibility to protect quality while controlling costs in patient care, will force physicians and the medical profession to do what no one really wants to do: be responsible for rationing care. Harry Schwartz put his finger right on it. Physicians are being placed in the role of "double agents" in health care. On the one hand, they want and are expected to be advocates for patients' interests, while on the other, they are finding themselves to be increasingly locked into being unhappy front-line agents for those whose goals are to achieve cost control.

It surely would not sit well with patients or the public if it became generally known that physicians were being maneuvered into becoming *de facto* double agents in patient care—and this very much against their will, their tradition, and their ethics, which have always put patients' interests first. As Schwartz suggests, perhaps it is time for physicians and organized medicine to begin to publicize to their patients and the public the fact that society itself is forcing physicians to become what amounts to double agents in patient care, and that this is not in the long-term interests of either patients or of society. If there is to be integrity and trust between doctor and patient, and this has always been a *sine qua non* in patient care, then a physician's first duty must be to a patient. Patients expect nothing less, and in the final analysis, society expects nothing less.

It would not only be a tragedy but a disaster for medicine if physicians and the medical profession were, by their own default, to become institutionalized as "double agents" in this nation's health care system. What is urgently needed now is for society, whether in the form of government, health insurance programs, or health care plans, to decide and make clear what they will pay for in health care and what they will not. This would free physicians to do what they should do and are trained to do—what they think is best for their patients—and do this within the framework of whatever resources society decides it can afford. This will not be easy to do, but the stakes for patients and the public are very high indeed.

There may, however, be a precedent worth some thought and study. As Dr Kitzhaber points out elsewhere in this issue, the state of Oregon has been one of the first to recognize and face this problem and has already begun to take some steps to deal with it. But there is as yet no widespread general awareness. The immediate need is for affirmative action by individual physicians with their patients and by the medical profession with society as a whole to publicize this issue and then to work with the payers of health care to find ways to separate the roles of church and state, so to speak, or in health care, the appropriate roles of physicians and society.

MSMW

What Do Stroke and Brain Trauma Have in Common?

WHILE MANY INVESTIGATIONS of acute central nervous system (CNS) injury are now emphasizing cellular mechanisms, improved patient care is still the ultimate goal of this research. An important underlying hypothesis is that populations of dysfunctioning neurons intermix with or surround areas of dead or dying neurons and that these cells, while vulnerable, may recover if the local environment can be altered from unfavorable to favorable. Studies of later recovery are moving in parallel where many investigators are also studying cellular events. Particularly interesting are those that reexamine the roles of regeneration and sprouting. These issues, specifically neuronal vulnerability after injury due to unfavorable environments, drugs that may alter these environments, and the role of axonal sprouting, are addressed in this issue of the JOURNAL by Becker and associates, "Brain Cellular Injury and Recovery—Horizons for Improving Medical Therapy in Stroke and Trauma."

The reader whose experience is outside the area of CNS injury may ask, why stroke and trauma? Although they account for the major proportion of CNS-related death and disability, the two conditions are distinctly different. While differences are obvious, however, there are at least two important reasons to consider and, further, to study them together. First, there are abundant data showing a coexistence of traumatic brain injury and events that can cause cerebral ischemia and hypoxia in a high proportion of head injury

cases. Second, important cellular events that cause secondary injury may be similar or even identical in both stroke and trauma. The animal models described by the authors, employing sequential injury—impact followed by ischemia—are, then, appropriate from both perspectives. Further, these models at least indirectly address the central hypothesis of increased neuronal vulnerability and secondary cell death after injury. They also define specific candidate mechanisms that may underlie this vulnerability and provide new directions for therapy.

Several published reports have demonstrated the coincidence of head injury and shock or hypoxia. For example, consider data from the pilot phase of the national Traumatic Coma Data Bank.[1] In that study, data were collected prospectively on almost 600 patients. Of these patients, 20% were considered hypoxic before admission, as defined by an initial partial arterial oxygen pressure of 60 torr or less, or by a history of apnea in those patients ventilated before their arterial blood gases were measured. Shock occurred in 33% of the population as defined by a systolic blood pressure of 90 torr or less before admission. Shock was associated with a 30% increase in the frequency of the outcome of brain death or vegetative state, while hypoxia was associated with about a 20% increase in this outcome. Additionally, in association with these insults there were relatively fewer patients who were considered to have a good outcome or to be only moderately disabled. These associations were statistically significant. Further, while there is, no doubt, an association between multisystem injury and either shock or hypoxia, a statistically significant association between multisystem trauma and outcome could not be found in this population. Data from the full-phase study, which includes information from more than 1,000 other patients, are currently undergoing analysis and also show the importance of early hypoxia and shock in patients with severe head injury.

What are the specific cellular mechanisms common to stroke and trauma that could cause increased neuronal vulnerability in secondary cell death? Those currently under the most intensive investigation and discussed by Becker and co-workers have a central theme of the release of substances from injured or dying cells, substances assumed toxic to nearby neurons spared by the initial precipitating event. These mechanisms include the accumulation of lactate and acidification of the local environment; cell-membrane breakdown, lipid peroxidation, and release of free radicals; and excessive release and accumulation of neurotoxins and excitatory synaptic transmitters, particularly glutamate. A recently published report from the Cornell group headed by Plum and Pulsinelli has provided important direct evidence that lactate in concentrations like those found after ischemia can cause neuronal death.[2] These investigators studied the histologic appearance of rat brains that had been subjected to microinjections of various concentrations of lactate. The idea that lactate or hydrogen ion is toxic to neurons now seems secure. In view of the controversies surrounding this idea, it is surprising that this experiment was not done earlier. Also recently reported and interesting are the findings of Pitts and associates.[3] They studied a sequential injury model using magnetic resonance spectroscopy. Pertinent to the hypothesis under consideration here was the finding that intracellular pH and high-energy phosphate concentrations (adenosine triphosphate and creatine phosphate) decreased when both injuries were combined—impact followed by hypoxia—to a much greater degree than when the two injuries were studied separately.

While the idea that glutamate in high concentrations may act as a neurotoxin is not new, compounds that would easily penetrate the blood-brain barrier and act as specific receptor (*N*-methyl-D-aspartate) antagonists have only recently become available, and one such compound, MK-801, has been shown in animal models of ischemia to have beneficial effects.[4] Of the three outlined mechanisms, the release of free radicals as a cause of secondary neuronal injury in animal models has been the most widely supported; data have been generated from many laboratories under a wide variety of conditions. The evidence is sufficiently impressive to warrant a clinical trial in the context of a multicenter study, and the United States Department of Defense has funded such a trial of superoxide dismutase in patients with missile injuries of the brain.

There has been considerable progress in studying these mechanisms, though their clinical importance must await further study. It is hoped that when indicated, they will be tested by multicenter, randomized trials. The clinical importance of cellular mechanisms in the animal studies of long-term recovery, particularly those indicating a possible role for axonal sprouting, will obviously be more difficult to evaluate. These findings are potentially the most exciting, however, and these kinds of experiments should be encouraged.

HOWARD M. EISENBERG, MD
Division of Neurosurgery
The University of Texas Medical Branch
Galveston, Texas

REFERENCES

1. Eisenberg HM, Cayard C, Papanicolaou AC, et al: The effects of three potentially preventable complications on outcome after severe head injury, chap 93, *In* Heidelberg V (Ed): Intracranial Pressure. New York, Springer-Verlag, 1983, pp 549-553

2. Kraig RP, Petito CK, Plum F, et al: Hydrogen ions kill brain at concentrations reached in ischemia. J Cereb Blood Flow Metab 1987; 7:379-386

3. Ishige N, Pitts LH, Pogliani L, et al: Effect of hypoxia on traumatic brain injury in rats: Pt 2. Changes in high energy phosphate metabolism. Neurosurgery 1987; 20:854-858

4. Kochhar A, Zivin JA, Lyden PD, et al: Glutamate antagonist therapy reduces neurologic deficits produced by focal central nervous system ischemia. Arch Neurol 1988; 45:148-153

Uncompensated Care

"MY JOB AS A PHYSICIAN is to serve as an agent of trust and to do anything appropriate to provide necessary medical care and not to serve as a rationeer of scarce resources on an individual basis." This statement of Dr Nancy Dickey, chair of the American Medical Association's Council on Ethical and Judicial Affairs, expressed the convictions of most physicians. None of us, as physicians and human beings, want to make our professional decisions for our patients primarily for fiscal reasons. We desire to give them quality care and to be their advocates when needed.

Society may change this. It may mandate us to render inadequate or improper services if the problems associated with uncompensated care are not solved. The growing crisis of caring for those who are uninsured, underinsured, and uninsurable, according to Dr John Kitzhaber, ". . . poses one of the most serious threats facing the medical profession today." It can result in a deterioration of health for a growing number of Americans, with very serious social and economic consequences.

Dr Kitzhaber, whose address to the California Medical Association House of Delegates at its 1988 annual meeting is